THE PATHOLOGICAL PRODROMES OF TABOPARESIS

SYPHILIS; MENINGITIS; THE GENESIS OF THE CRANIAL NERVE IMPLICATIONS

BY

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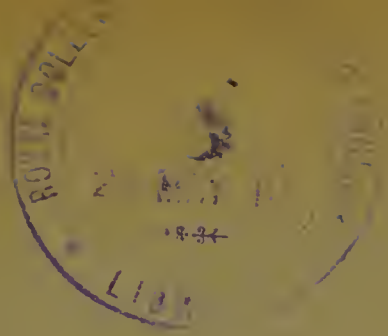
WASHINGTON, D. C.

Reprinted from the MEDICAL RECORD
February 5, 1910.

WILLIAM WOOD & COMPANY
NEW YORK



THE PATHOLOGICAL PRODROMES OF TABOPARESIS.*

SYPHILIS; MENINGITIS; THE GENESIS OF THE CRANIAL NERVE IMPLICATIONS.

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ONE of the main contentions against the ideas of Nageotte as to the pathogenesis of tabes dorsalis has been the supposed unessentialness of the chronic meningitis he invoked as the necessary antecedent of locomotor ataxia. As to his 11 cases all showing marked meningeal exudates, the objection is made that they were really exceptions; for other observers had failed to find lesions in the meninges. Nageotte believed that an incomplete examination was the cause of this; but, in turn, it was objected that if his theory were correct, meningeal inflammation should have been found around the cranial

*Through the kindness of Dr. Clovis Vincent, the writer is enabled to place before American readers the results of an investigation Dr. Vincent has been pursuing during the last five years in the clinics of the Parisian Hospitals La Pitié, La Salpétrière, and St. Louis, and in the laboratories of Babinski and Nageotte, Raymond, and Chauffard. The reader will see that the results of Dr. Vincent's research afford still further corroboration of the ideas so long held by Babinski and Nageotte that all the symptoms of taboparesis ensue upon a chronic meningitis of syphilitic nature. The data here expounded clearly show the untenability of the dystrophic theory of taboparesis.

nerves, which are so often implicated symptomatically early in tabetics.

Very few of Nageotte's opponents go so far as to deny that persistent lymphocytosis really indicates meningitis; and it is pretty generally conceded to be pathognomonic of meningeal irritation at least. We need not quarrel about terms, for the commencement of the inflammatory process may be designated by

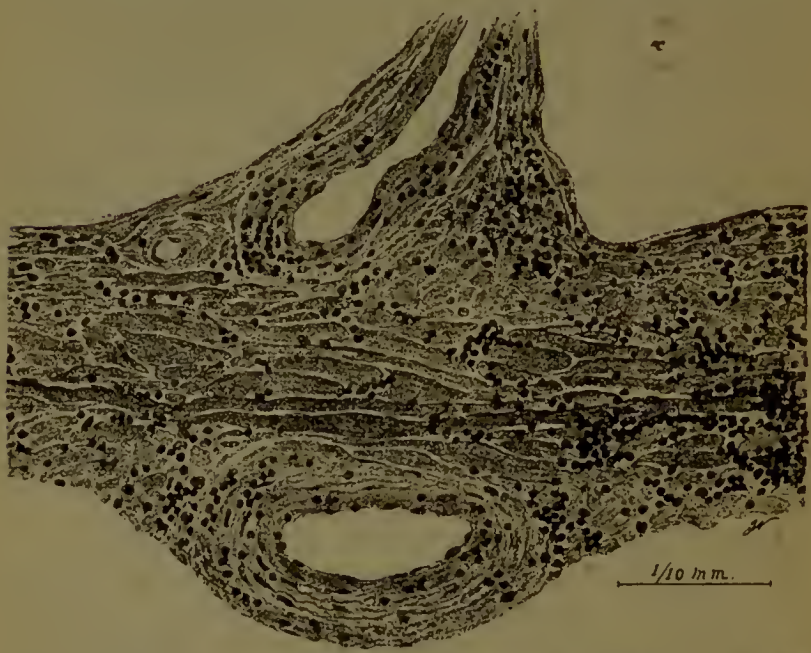


FIG. 1.—Showing the type of meningitis in taboparesis. It shows the pia mater over the posterior region of the spinal cord at a dorsal segment magnified 205 diameters and stained with haematoxylin and eosin. Note thickened connective tissue and the infiltration of lymphocytes and epithelioid cells; also a thickened blood vessel entering the spinal cord along a septum. (From Nageotte.)

the term irritation; hence the difficulty is merely one of stage, measured by degree. Moreover, the appearance revealed under the microscope, after the death of the patient who has shown persistent

lymphocytosis, of considerable meningeal infiltration with small round cells as well as with commencing and even complete organization of chronic inflammation proves that lymphocytosis does not occur by a mere multiplication in the blood of the white cells, as in leukemia.

I am now speaking of the abundant lymphocytoses so characteristic of syphilis. The lymphocytosis sometimes found during mumps and even that occurring so often in herpes zoster are less abund-

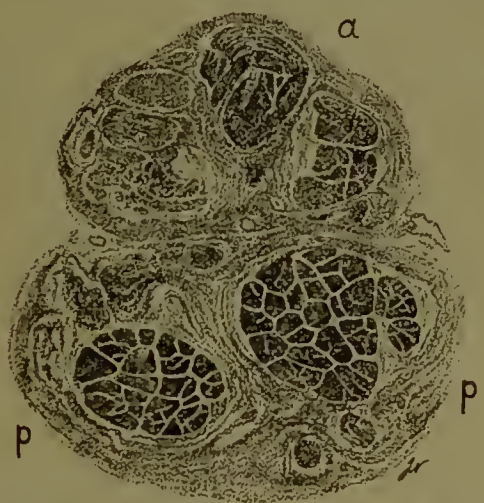


FIG. 2.—Extensive and marked perineuritis which has not yet implicated the fibers of the posterior root bundles (p p), but has seriously invaded those of the anterior root (a). Many of these latter are quite degenerated, shown by their disorganization and failure to stain. A radicular nerve of a taboparetic, the second sacral. X 10. (From Nageotte.)

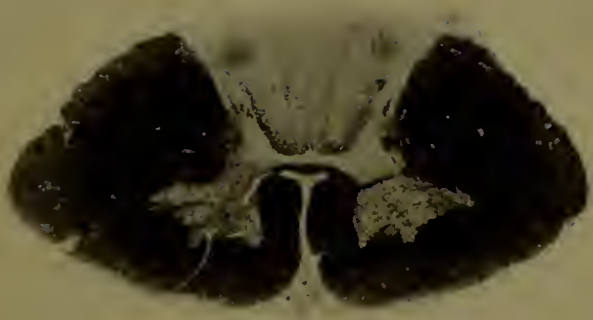
ant and disappear quite rapidly. For the purpose of this discussion, we are dealing only with such marked exudates as almost crowd the field; that is to say, occurring in blocks or chains, and indicative of severe meningeal reaction. Hence the demonstration does not depend upon the refined and diffi-

cult laboratory methods, and is within the reach of any practitioner.

The cells in the syphilitic meningitis differ also in kind, being surrounded by a narrow zone which easily colors with orange. Moreover, the polynuclear neutrophile cells may also occur; but the most characteristic cells are the basophile young plasma cells, which, it must be remembered, are very unusual in the really normal connective tissue, and are pathognomonic of chronic morbid reaction. The kidney-shaped mononuclears and the active macrophages and the eosinophiles may also be present.

Now, although such appearance always occurs in syphilis of the nervous system, yet we cannot affirm that they are syphilitic in nature until we find the *Treponema pallidum,* or find some antibody to it. The reaction devised by Wassermann is not at present believed to be really specific in the sense in which Bordet and Gengou first devised the principle. For absolute rigor we must therefore fall back upon the law of invariable succession.

A most important advance in our knowledge of this succession has been made by the researches of Vincent. He has had occasion to puncture the arachnoid sac of a large number of patients with and without syphilitic history, the latter sometimes showing only a single symptom, such as paralysis of the third, sixth, seventh, or eighth nerve. He has also punctured the subarachnoid cavity just after the onset of symptoms. In all of these he has found considerable lymphocytosis, and it seems absurd to suppose that a reaction of this nature is not indicative of chronic meningitis antedating for a considerable period the nervous symptoms for which the patient consulted, which had lasted only a few days in some instances.



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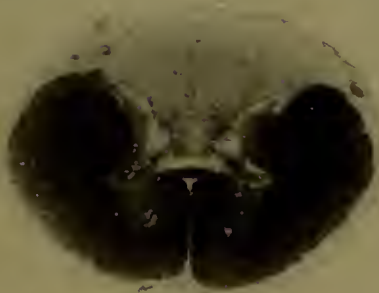

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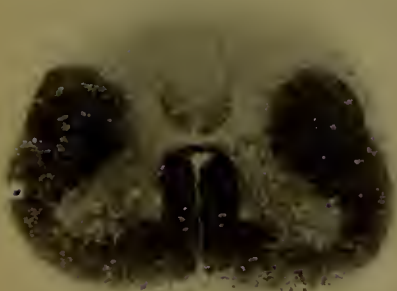

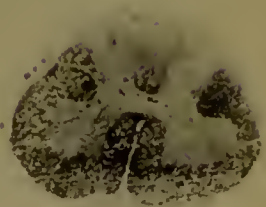

C D

FIG. 3.—Sections of spinal cord of a tabetic (Case IX), showing a degeneration of the posterior columns of Goll and Burdach along their whole extent with conservation of the cornu-commisural zone, and some other endogenous fibers only (Weigert), A, cervical region; B, lower dorsal; C, fourth lumbar segment; D, sacral region.

Another powerful argument is the fact that considerable lymphoid infiltration has been found in the connective tissue surrounding nerves whose functions were apparently unaffected during life, and similarly the pia mater also presented extensive exudates where no mental symptoms had appeared.

But for absolute proof one would require the most systematic punctures of all syphilitics long before they present the tertiary nervous symptoms; and Vincent has reported five cases of this kind. In all the first three patients, nerve symptoms have already shown themselves; in one, diffuse meningo-encephalitis; in the second, a double hemiplegia; in the third, a neuritis of both eighth nerves.

Case I.—A man, 42, in April, 1898, felt tired and had occasional headache. Treated for syphilis twelve years before. There were no signs of disease of the nervous system, except that the right pupil was smaller than the left, and contracted less freely and extensively to light, both direct and consensual. It was this sign which compelled the lumbar puncture, although he had no change in speech, loss of memory, disorientation, or change of humor, and had been at work up to the day before. Enormous lymphocytosis was found. The patient was again seen six months later. He was emaciated, stuttered, could not repeat correctly the test phrases, his tongue and face trembled, he was untidy, forgetful, not being able to find his way home; in short, all psychic and somatic signs pointing to general paralysis. The right pupil reflex, however, was in the same state as at the first examination. Thus, lymphocytosis was a sign indicating chronic meningitis preceding general paralysis.

Case II.—Man, 52. Syphilis, 1892. Treated three months by injection, and two years and one-

half with mercurial pills; later potassium iodide. During the secondary period he had had articular pains except when heated by movement. Later a severe permanent headache, diurnal in type (he worked at night as a baker). Slight alcoholic gastritis had troubled him, but was easily cured in 1898. The joint and head pains persisted, so the meninges were punctured, and the liquid was found

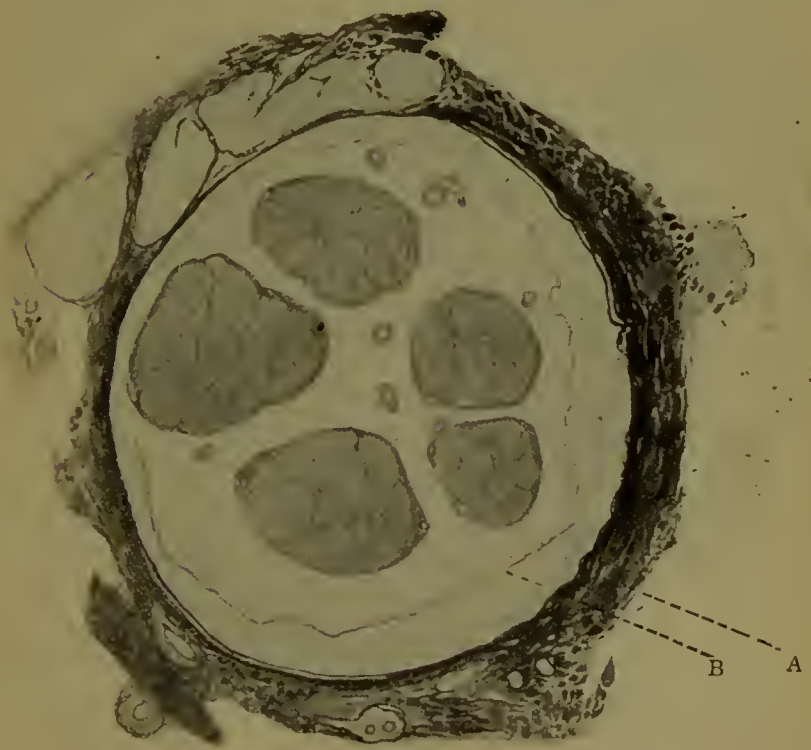

FIG. 4.—Transverse section of normal hypoglossal nerve after its fascicles have collected into the common meningeal sheath which surrounds them before they enter the anterior condyloid foramen. This situation is the homologue of the "radicular zone" of the spinal roots. Note the thin single layer of arachnoid matter and the equality of size of the nerve fibers and medullary rings which are alongside one another (Van Gieson stain). A, dural sheath; B, arachnoid sheath.

to abound in both lymphocytes and polynuclears. Two further punctures revealed the same lymphocytosis, but no objective nerve symptoms were present, until one night in October, while at work, he had a fit, followed by a complete paralysis of the face and all four limbs. He could not even swallow. Liquids were given through the nose. He had ptosis, saw double, and the tongue was clumsy. On

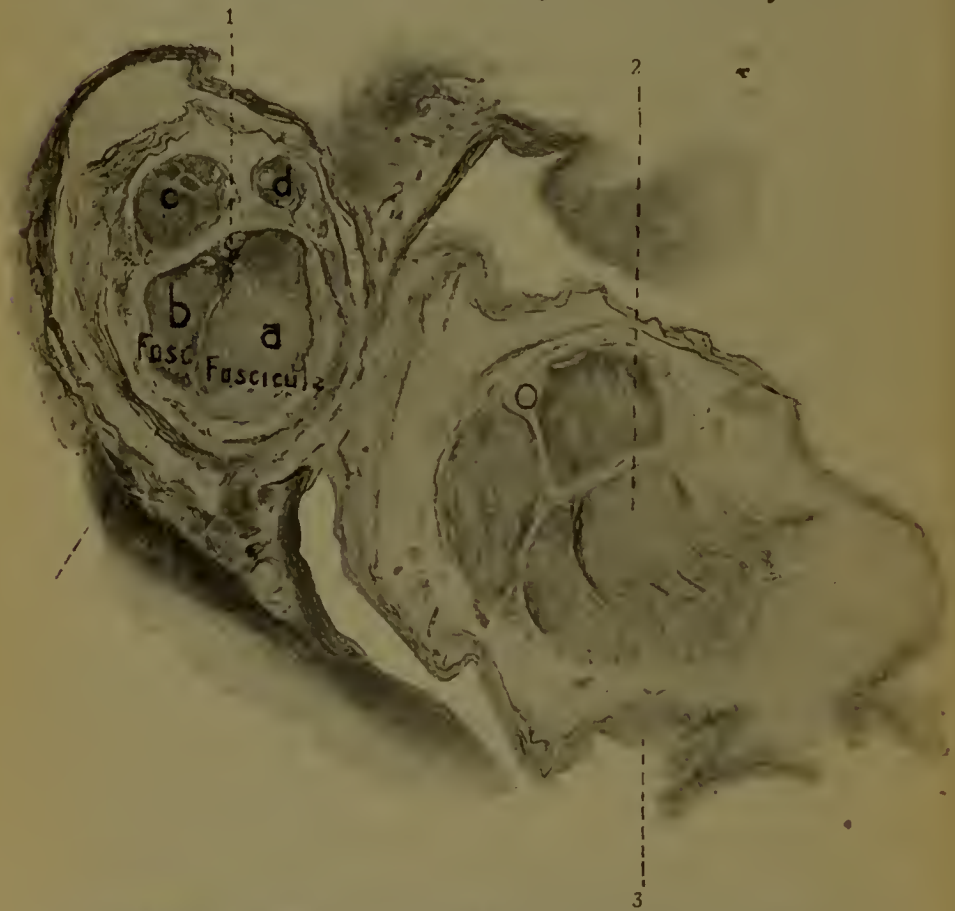

FIG. 5.—Similar section of hypoglossal nerve of this patient, see Case IX, showing thickening of the arachnoid. Round fasciculus (1) and also on the inferior aspect of fasciculus (2), connective tissue areolæ are separating the nerve fibers into groups (Van Gieson stain).

mercurial treatment, he began to walk and swallow. Three months later the left deep reflexes were greater than those on the right, which presented the platysma sign. The association movement of the thigh and pelvis was impaired on the right side. Swallowing and speaking were still affected. There was no irideplegia, but lymphocytes were still abun-

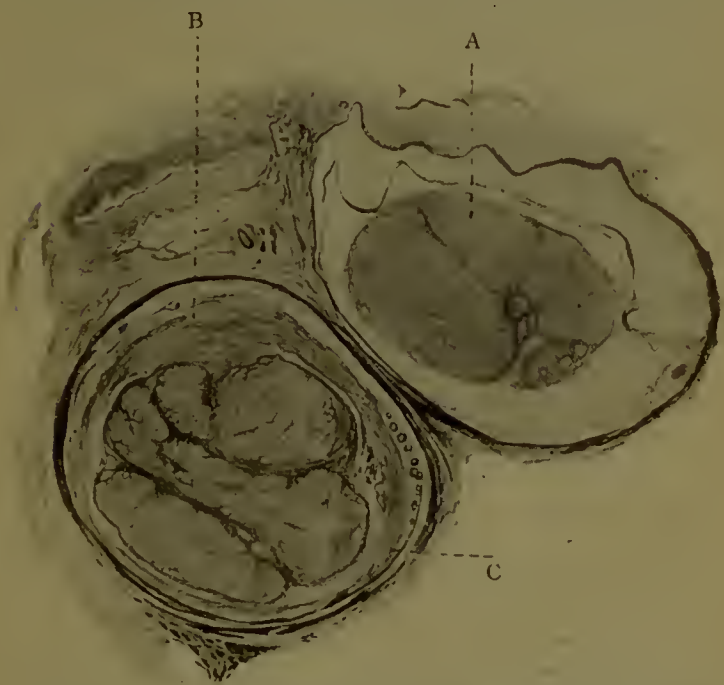

FIG. 6.—Section of same nerve nearer its exit. Fasciculus A now shows only a slight endoneuritis. Its arachnoid sheath is now almost normal in thickness; while fasciculus B shows intense endoneuritis and a greatly thickened arachnoid (Van Gieson stain).

dant. There was apparently a partial thrombosis of the basilar or its branches, for the pyramidal and geniculate tracts were implicated, as well as the cranial nerves. Thus for at least a year chronic syphilitic meningitis, as evidenced by lymphocytosis,

had been present before cerebrospinal syphilis declared itself symptomatically.

Case III.—The third case of this series was that of a child aged 8. After a chancre of the lip in March, 1897, and without any secondary symptoms, beginning in May, she contracted in turn a gumma of the clavicle, and an interstitial keratitis. It is probable, therefore, that even then meningitis was present, for Vincent has never seen a case of interstitial keratitis unaccompanied at one time or another with lymphocytosis. But the girl recovered under mercurial treatment until in March, 1898, she suddenly became deaf. Tinnitus was also present; and on examination, the labyrinthine origin of the trouble was evident, though there was no vertigo nor other sign of organic nervous disease. In spite of the treatment, the deafness increased, and in July was complete. Enormous lymphocytosis was present during all this period, and the Wassermann reaction was positive. Thus again a lesion of the nervous system appears as the third stage of a process of which the first was syphilis, and the second meningitis.

Case IV.—In the next two cases this third stage has not yet occurred. The first is that of a man of 35, having had syphilis in 1901. He received inunctions for the first two years, taking iodide between the series. He then ceased treatment for two years, after which he recommenced on account of three miscarriages by his wife in 1903, 1904, and 1905, the last one a living child with hereditary syphilis. It seems evident then that in spite of the treatment there has been no interruption in the disease between the initial chancre and the results following; for in 1907 a lumbar puncture showed abundant lymphocytosis. Yet all his nervous func-

tions were unimpaired with the exception of a certain instability in face and speech which he has always had, but he continues at work, although he has had some trouble with his wife, and appears a little eccentric.

CASE V.—A man, 24, had syphilis in 1904, without secondaries. He commenced using gray oil

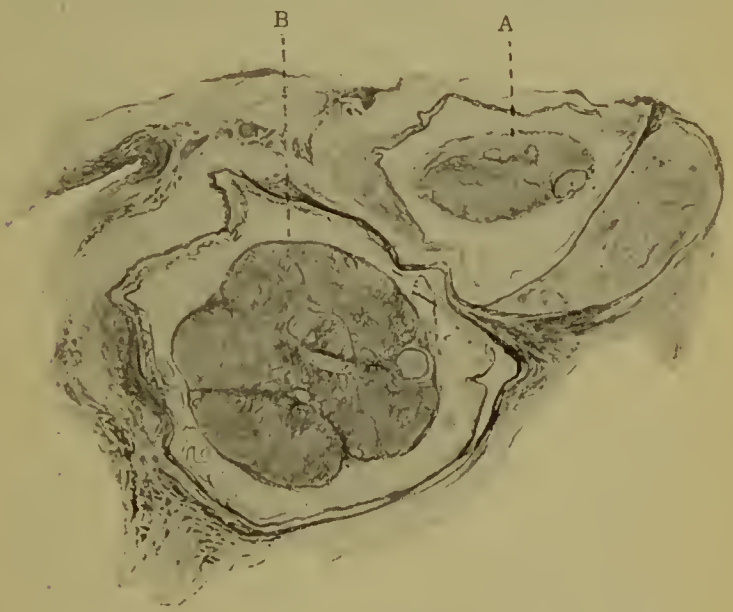

FIG. 7.—Still lower, fasciculus A is much atrophied; fasciculus B still shows endoneuritis, but little arachnoiditis now remains. These three figures clearly show in these two fascicules, an inflammatory area which arises, increases, shows a maximum and decreases; it deserves the name of transverse radicular neuritis (Van Gieson stain).

from the time he saw the chancre, treating himself, but soon ceased on account of the pain, and substituted a daily dose of 10 centigrams protiodide of mercury. This he continued for three years. Then, believing himself cured, he married. However, 27 months after the chancre headache began and lasted

almost continuously for 8 months. Though worse at night, it was thought to be in turn influenzal, neurasthenic, and neuralgic. On examination, no signs of organic nervous disease were discovered, but punctures showed a tremendous lymphocytosis, such as appears in the most active cases of general paralysis. Indeed, the diagnosis could have been made with the naked eye, the liquid showing a peculiar opalescence characteristic of an abundance of cells. In addition to the usual cells there were some polynuclear and eosinophile, and also some plasma cells. He was at once treated by intravenous injections of mercuric cyanide every other day to the number of thirty in all. The headache quickly disappeared, but he was then given six injections of gray oil and four series of six injections of emulsified calomel. He might have been considered as cured but for the appearance of a pharnygeal gumma 6 months later, and but for the persistence of the intense meningitis shown by the lymphocytosis.

The future of these patients will contribute to the solution of a medical problem of great importance, for if it is ever shown that specific treatment can remove chronic meningitis, it will be necessary to puncture systematically all cases of syphilis, so that the meningeal trouble may be detected in its incipiency and before causing irreparable lesions of the nervous system.

But it had been known since the researches of Ravaut in 1903 that meningitis often occurs in the secondary period, and is sometimes even followed by lesions of the nervous system, facial palsy and neuralgia being the most common, so that it is very important to know whether the tertiary meningitis really has been preceded by a latent secondary one.

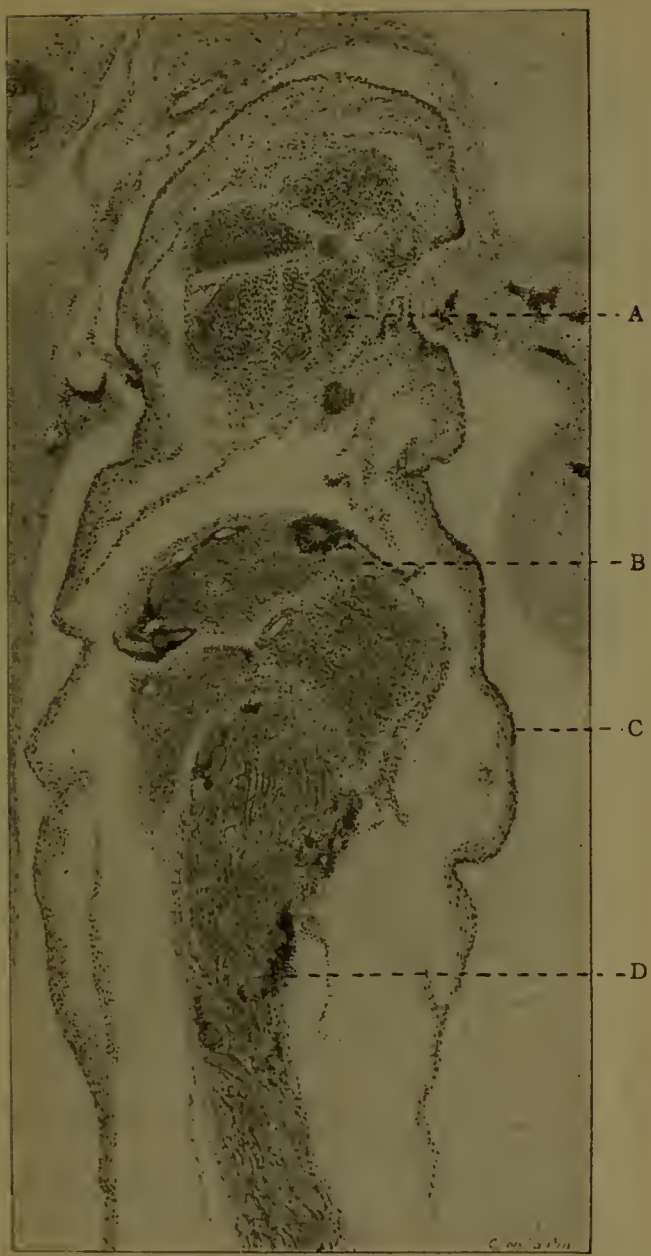

FIG. 8.—Section just above that of Fig. 5 (B is cut longitudinally as it bends), stained with hematein and eosin to show the infiltration of round cells and the progressive thickening of the arachnoid as it descends to embrace the nerves; note the granulomata. C, artery and vein; D, arachnoid, growing thicker as it ascends.

The following cases are instructive in this regard:

Case VI.—A single woman of 30 had a chancre in 1900. Facial paralysis six months later. In December, 1902, only a slight spasm remained. At that time considerable lymphocytosis was present.

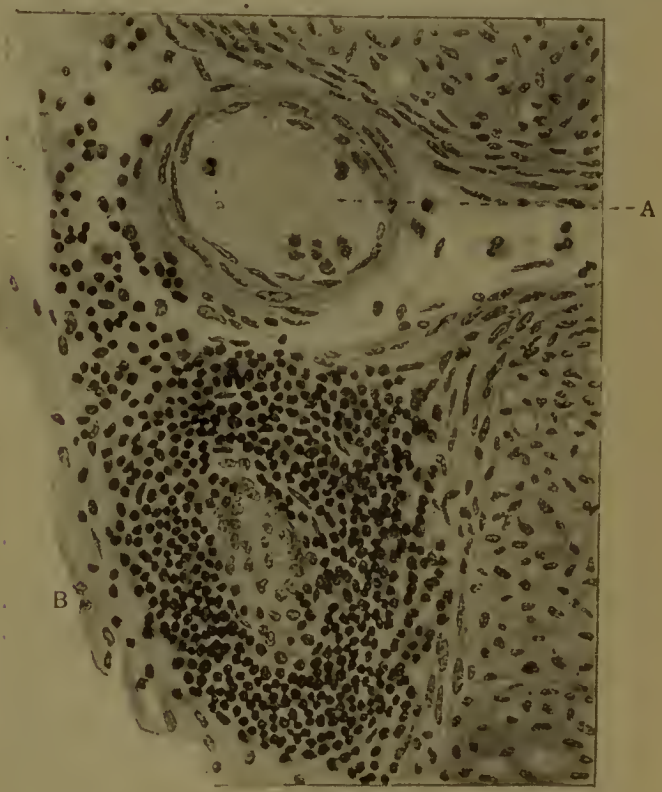

Fig. 9.—A perivenous granuloma of Fig. 8 under a high power; note the commencing organization of the cellular elements. A, artery; B, vein.

After four series of gray oil injections, the lymphocystosis was still present. It was not until four years had elapsed that there was any considerable amelioration. There were only five or six lymphocytes in each field, and this improvement

still continues; so that she may be regarded as out of danger for the time being at least.

Case VII.—A single woman, 28; had roseola in 1901, and numerous secondaries. In 1903 trigeminal neuralgia, more especially frontal, which was

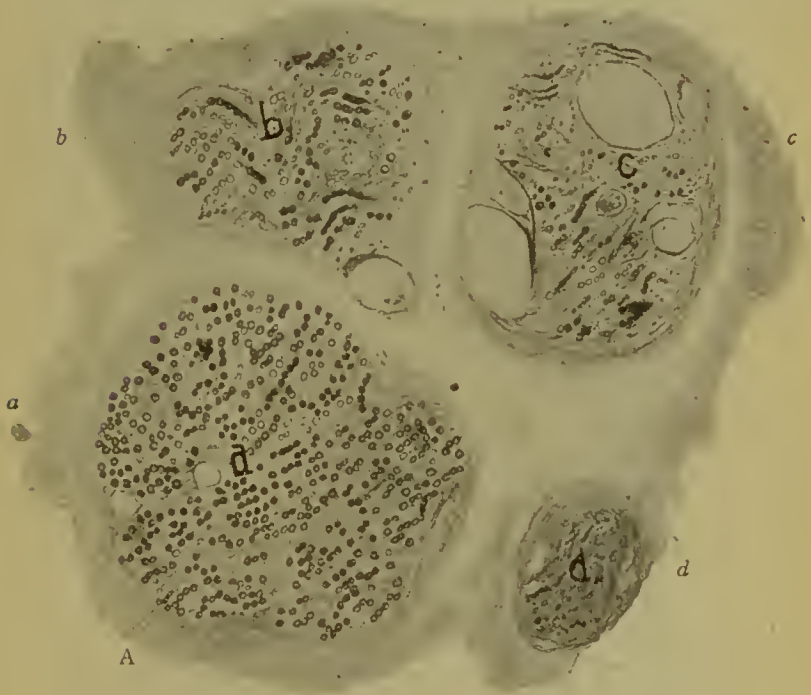

Fig. 10.—Fascicule A (Azoulay stain); note in *a* some fibers uncolored or small, and the progressive diminution of colored fibers, in *b*, *c*, and *d*.

followed by a double hemorrhagic retinitis. An enormous lymphocytosis was present. A year later, right hemiplegia and aphasia appeared, the symptoms of which were practically well a year after. She was punctured periodically until October, 1908, and always showed an enormous quantity of cellu-

lar elements and albumin in the arachnoid fluid.

It seems very probable, therefore, that syphilis of the nervous system sometimes may be traced directly back to the general infection following the chancre,

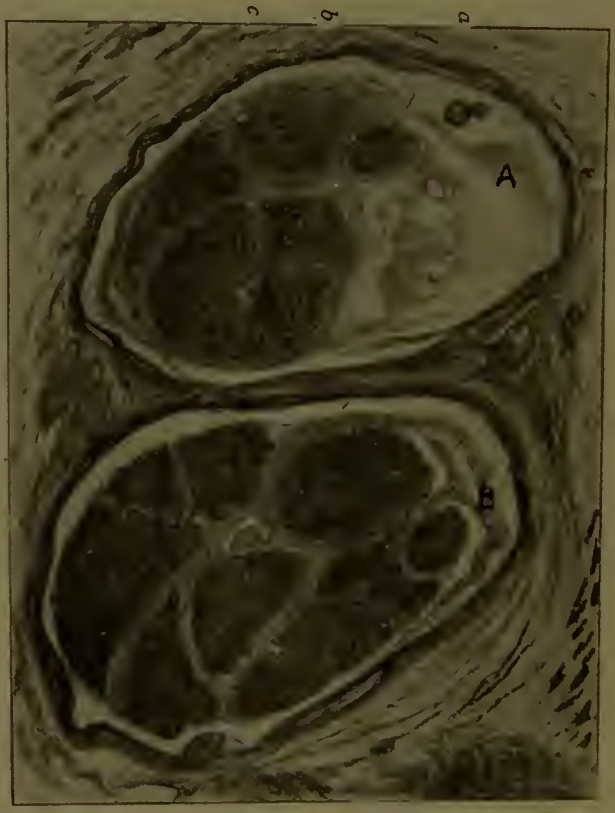

FIG. 11.—Part of the pneumogastric nerve of same Case IX, showing endoneuritis and arachnoid hyperplasia; very little myeline remains in fascicles *a*, *b* and *c* (Von Gieson).

and that chronic meningitis is merely the persistent state of the secondary period; for it must be remembered that nervous accidents may occur in the secondary period, and that examination of these reveals always a lymphocytosis, as in the case de-

scribed by Sézary (Soc. de Biologie, 1908, April). It was a man of 40, who a month after the chancre showed general psoriasis, and forty days later, hemiplegia, aphasia, and death. Two lumbar punctures at a month's interval showed abundant lymphocytosis, the first having been made before the nervous symptoms had appeared.

Case VIII.—Another case is that of Dutheil (*Thèse de Paris*, 1909). It was a man of 43, who five weeks after the chancre had severe papulotubercular syphilides, showing lymphocytosis. Mercury had to be given up on account of stomatitis. Twenty-six days after he fell into a comatose state and could hardly swallow. Eight days later right hemiplegia, with Babinski sign and aphasia, and death so rapidly that no cerebral softening was found post mortem.

In both cases the same striking sequence is present of syphilitic meningitis and organic nervous disease. The rapid succession shows the relation all the more strikingly, and thus there would not appear to be the essential difference formerly supposed between secondary and tertiary manifestations.

The sceptic, however, will still require pathological confirmation of this thesis, and, although this has been given as regards the spinal root by Nageotte's researches, confirmation with regard to the cranial nerves has not been presented. Now, however, the case which follows furnishes irrefragible proof of each of the foregoing points.

Case IX.—A man of 38 entered the Salpétrière in 1907 for weakness of the lower limbs. He had syphilis in 1891, inadequately treated. He drank a great deal. In 1904 he began to have vertiginous turns and feelings of suffocation in the night. Later

began girdle pains, which he thought were colic. On examination he was failing, emaciated, and appeared 60 years of age. It was a typical case of advanced tabes, with abolition of all deep reflexes and a conservation of those of the skin. He was

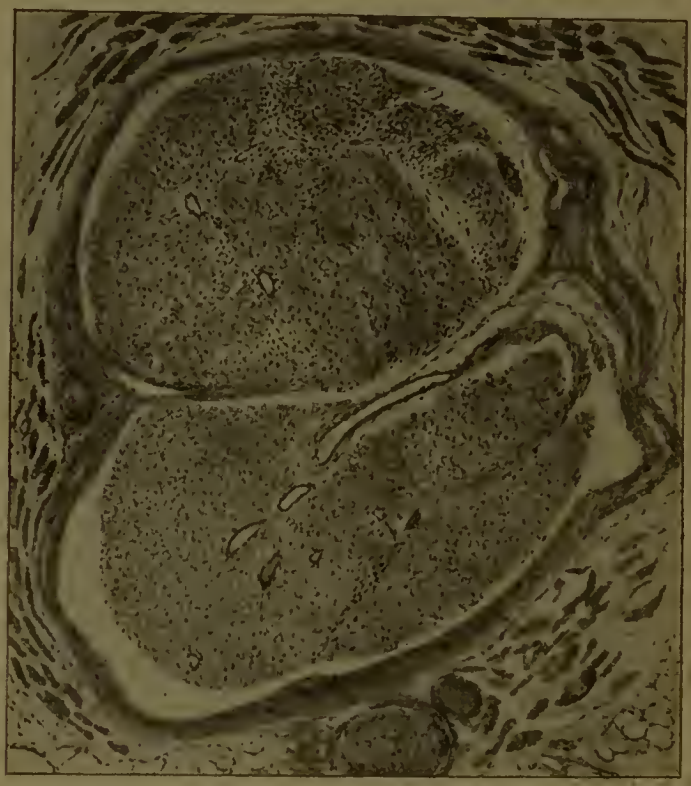

FIG. 12.—The same with hematin-eosin showing round-cell infiltration of nerve and meninges.

very ataxic. The muscles were diffusely atrophied, especially in the peroneal region, and power was diminished in the pelvic girdle; but a reaction of degeneration occurred only in the right extensor communis digitorum. Sensory symptoms consisted of lightning pains, complete loss of sense of attitude

(less marked in the arms), slight hypoalgesia, while the sense of touch was not impaired. There was incontinence of urine. The right pupil was larger than the left, and neither reacted to light nor accommodation. There was intermittent diplopia, due to paresis of the right internal rectus. The fundus showed a lesion. There were attacks of suffocation beginning suddenly, with cold sweats, during which occurred asphyxia, and sometimes loss of consciousness. He had also laryngeal crises, and there was paralysis of the abductors. There were no other cranial nerve symptoms.

The intelligence seemed intact, as he read the papers each day and spoke reasonably to his wife, advised the patient in the neighboring bed, and was very grateful for the care he received. This is particularly striking in view of the fact that the autopsy showed a diffuse cortical meningeal involvment; but it was merely the first stage, for the process had not invaded the pial septa; and there was no encephalitis to constitute the picture of the general paralytic brain. The process, however, had penetrated the medulla, as will be seen.

Autopsy.—The whole neuraxis showed an extensive meningitis. There was a very opalescent aspect of the pia mater, especially along the vessels on the convexity. At the base of the brain, on the contrary, an enormously thickened layer of arachnoid clothed the chiasm in the interpeduncular space, and extended along the optic nerves. This was even more marked over the pons, a fibrous mat being furrowed by the facial and auditory nerves. The appearance of the cerebellum was like that of the cord. There was extensive meningitis along the posterior surface of the dorsal cord. There was a trimeningeal symphysis extending to

the lumbar enlargement. No lesion of the nerve centers could be observed with the naked eye. Thus we see basillar meningitis, the first stage of general paralysis, and meningitis of developing tabes, placed before us by the same patient.

Microscopical Appearances.—The cord showed the (Fig. 3) typical appearances of tabes, with the almost complete integrity of the endogenous tracts and short exogenous fibers. The tabes is not only lumbarsacral, but also dorsal and cervical. The transverse radiculitis was present in every respect like that described by Nageotte. The cortex showed lymphocyte infiltrations of the pia matter with endoperiarteritis; but the intracerebral vessels were free, and the tangential fibers and pyramidal cells were not implicated.

The left hypoglossal and right pneumogastric were damaged at the autopsy; but, although there had been no clinical evidence of its implication, in spite of the repeated careful examination of the face and of the tongue, the right hypoglossus showed all the stages between complete health and entire destruction. The absence of clinical symptoms could be accounted for in view of the fact that only about 1-16 of the fibers were atrophied. The loss of function of the muscles supplied by these was masked by the presence of the remaining healthy fibers; for there is great interlacement of the muscle fibers of the tongue. In the oculomotor on the contrary the muscles are separated, and have to be in a state of equilibrium to their antagonists. A very small lesion will disturb the exactness of this adjustment; and a diplopia appears at a stage when mere inspection could not detect the muscular disability.

As the chain of threads of the hypoglossus emerges from its sulcus in the medulla, they normally converge and become the nerve. Its constituents become surrounded by meninges which form a funnel-shaped canal. There may be one, two, or more of these, each containing several fasciculi, which assume polygonal form from their contact with one another. Between the fasiculi is a slender network of arachnoid, containing a few vessels. The whole is surrounded at a distance by the perietal arachnoid, which again is separated from the dural sheath which surrounds this; there is very little connective tissue between, and finally the individual nerve fibers are pressed one against the other, almost free from connective tissue, which is only visible with a very high power. See figure 1 of normal hypoglossal nerve.

On comparing this with the nerve from this patient (Fig. 2), one sees an enormous increase of connective tissue within and around the fasciculi-endoneuritis. Moreover, the proliferation of the perifascicular arachnoid has joined it at one spot with the thickened arachnoid sheath, constituting perineuritis. The activity of the lesion is shown by the lymphoid character of the exudates.

In this patient there were four hypoglossal fasciculi, two of them large; and it is very interesting to study the difference of level at which these were respectively implicated in the meningeal process. The most striking fact of all, however, was that the inflammatory processes entirely ceased before the two fasciculi formed the hypoglossal trunk. That is to say, its localization constituted the complete homologue of Nageotte's radicular sac in the spinal nerves. It began even before the common arachnodural sheath, which also surrounded the

filaments in a common fasciculus. It was shown by a thickening of the sheath around them, scarcely visible, and also by vascular filaments. The smaller fasciculi also showed this appearance along their nerve fibers.

A little lower down (Fig. 3*a*) the arachnoid gutter was enormously thickened, but inflammation had not yet attained its height within the fasciculi, although the nerve itself was five times its normal thickness. This progressive swelling of the perifasicular sheath is one of the most instructive facts of the whole research in that it explains why a pathological diffusive-process is, symptomatically speaking, revealed by so few definite signs. The reason why the lesion has so long escaped notice now becomes clear; for it is confined to the first few tenths of a millimeter of the sheath of the nerve. The foregoing figures show how the intensity of the lesion progressively diminishes from this point; so that four millimetres lower, the meningitis practically disappears, but its effect upon the nerve fibers is shown by their progressive disappearance, which constitutes an "atrophic" sclerosis. In fasciculus *b*, on the contrary, the atrophy was much less marked, although the meningeal lesion was as great as that of fasciculus *a*. This illustrates the rise and fall in intensity.

The facts shown by the Van Gieson stain are, however, merely quantitative. Still more striking is the picture shown when the tissue is stained by hematein-eosin-rose, which is not designed to show the collagen of adult connective tissue, but reveals the nuclei of active inflammatory cells. The nerve fiber bundles show a tremendous infiltration of small round cells and fibroblasts. The chronicity of the process is shown by the fewness of the poly-

nuclears and erythrocytes, and this is confirmed by the thickness of the arterioles and the obstruction of the lumen by the proliferation of the endothelium. Moreover, lymphoid nodules are here and there seen, especially around the veins. These appearances show that they are still in evolution. The fibroblastic organization of intervening connective tissue is shown by the adhesion formed between the arachnoid and the several bundles, which had also extended even beyond to the duramater, which itself is inflamed.

The nerve fiber alterations are clearly shown by the methods of Weigert and d'Azoulay. The normal hypoglossus is like an anterior root in that it is composed of large nerve fibers nearly equal in circumference and almost touching one another. On impregnation with the osmotannic process, bundle *a* of the patient showed, even under the low power, to what a different degree fasciculi in the same section were implicated; thus *a* appeared nearly normal; while *b, c,* and *d* showed many dwarfed fibers. But the high power showed even in *a* that the fibers were abnormally separated one from the other; so that, although the fibers themselves were not yet attacked, the inflammation had already penetrated the nerve. Some fibers, though not entirely normal, had scarcely taken the stain. This seemed to be the first indication that they were attacked. Normal and abnormal seemed mixed without plan, although sometimes the diseased occurred in bunches. Lower down, *a* showed the same appearance as in the preceding section was shown by *b, c,* and *d.* That the appearances are not due to the technique is shown by the normal aspect of the normal fibers in the same fasciculus. It is quite clear that the maximum damage to the

nerve fibers occurs as they are traversing the inflammatory area in the meningeal sheath. It might be objected that this nerve degeneration was due to disease of the hypoglossal nucleus in the bulb; but 250 sections of the medulla failed to show any decolorized hypoglossal fibers or central softening. The meningitis was purely peribulbar, and the hypoglossal was intact within the metencephalon.

The predominant feature of the lesions of the nerve fibers is the demyelinization without loss of the cylindraxis. The loss of the myeline may extend both above and below the inflammatory focus. It may even extend to the periphery of the nerve. Unless reagents are employed which color either the axis cylinder or the degenerated myeline, one may conclude that a nerve is completely degenerated and wonder that its functions could have been maintained during life. The Weigert method shows neither the axis cylinder nor degenerated myeline; so that when conclusions are drawn from it alone, they may be fallacious. The conservation of the axones, for a time at least, explains the long continuance of the functions in nerves completely engulfed by a meningitic focus, and also why certain muscles sometimes escape, while others are implicated. Moreover, it is evident that there may occur alterations of the structure capable of modifying function and yet indistinguishable by our present technique from similar neighboring modifications of structure of sufficient intensity to interfere with function. And it is evident that if two neighboring fasciculi of the same nerve passing through the same diseased focus can be so different in their sensibility to reagents as well as clinically, still less wonderful must be the difference of the effect of the lesion in two nerves so widely

separated as the anterior and posterior spinal groups.

The pneumogastric nerves were examined with the same technique as the hypoglossal; but in addition, the peripheral portions, including the two recurrent laryngeal nerves, were studied. The irregular course of the filaments forming the vagus, and their enlacement with those of the ninth and eleventh nerves made them difficult to follow. Besides, there are two ganglia on the nerve, the first, the jugular, being very far from the subarachnoid portion of the nerve. In spite of these difficulties, the preparation shows (Fig. 11 and 12) similar lesions to those of the hypoglossus. The maximum of the endoneural and perineural inflammation occurs at the point where the arachnoid is reflected upon the nerve filaments. The arteritis is also abundant, and a definite exudate in course of organization is also found at the bottom of the arachnoid funnel.

The vagus normally contains many small fibers, which make it difficult to say that any have been stricken by the disease. It is clear, however, that the pale and small fibers are more numerous in the inflammatory focus. Moreover, at this point they are scattered; whereas in the normal nerve, they are close together. It is safe to conclude, therefore, that the vagus presents typical radiculitis.

The recurrent laryngeal nerve normally contains one fasciculus almost entirely composed of thick fibers. Its other fasciculi contain about one-third of slender fibers. In this case, osmic acid shows in two small fasciculi of the nerve, only from 15 to 20 thick fibers, whereas the normal nerve contains from 50 to 60. Blocks of connective tissue, some of them containing a small spot of myeline, separate the fibers of the diseased nerve. In the thick

fasciculus, which should normally consist of large fibers, slender fibers now preponderate. With the methods of Marchi, Weigert, and Azoulay only a few fibers color, and these are all large ones. It is clear, then, that there is great diminution of the fibers and that the small ones seen are abnormal. But Van Gieson's method shows a large number of intact axones and sheaths of Schwann; so that the lesion has not produced complete secondary degeneration, but only an extensive demyelinization and the loss of a certain number of large fibers. It would seem that the loss of these corresponds with the loss of the thick fibers in the fasciculus of the trunk in which the thick fibers had been destroyed to such an extent by the inflammatory lesion.

Medulla.—The nucleus ambiguus was examined from the acoustic tubercle to beyond the middle of the bulb. There was neither diminution nor atrophy of the cells on either side. The dorsal myeline was equally normal. The Weigert method showed that the axones were healthy while traversing the bulb. It is therefore clear that the laryngeal symptoms of this patient were due to a focus of transverse radicular meningitis.

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